vital importance than almost any other, and that it is, above all, the specialty of research.

And it should be remembered, also, that we are not a local, but a national Association, with natural functions of the nature of encouraging broad and liberal enterprises of education and investigation, and not merely a collection of workers, bent only on prosecuting our individual aims, and with no bond of union but our yearly meetings.

REPORT OF THE TREASURER.

Dr. Græme M. Hammond, of New York, Secretary and Treasurer, presented the Annual Report, which, on motion, was accepted. There was a balance in the treasury of $217.60.

SCIENTIFIC COMMUNICATIONS.

Dr. Philip C. Knapp, of Boston, read a paper on Nervous Affections following Brain Injury ("Concussion of the Spine," "Railway Spine," and "Railway Brain").

(For paper see October number.

Dr. Isaac Ott read a paper entitled

HEAT CENTRES IN MAN.

In this abstract I will give only the principal points of my paper on this subject.

There have been localized in the lower animals six centres whose injury causes increased temperature. The cruciate about the Rolandic fissure, the sylvian at the junction of the supra, and post-sylvian fissure; the caudate nucleus, the tissues about the corpus striatum, a point between the optic thalamus near the median line ; and the antero inner end of the optic thalamus. These centres have their own laws, which serve to distinguish one from the other. Dr. W. Hale White[1] has published a paper containing a number of collected cases bearing on this subject, and I have mainly used them to support the theory of localized heat-centres in man. He has ruled out all cases where the cause is not purely nervous.

In support of a heat-centre about the Rolandic fissure, there are two cases of injury in this region, which were followed by a temperature of 104° F. There is also a case of softening of the brain in the neighborhood of the Rolandic fissure, with a temperature of 105° F. reported.

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[1] Guy's Hospital Reports, 1884.

Corpus Striatum.—There are recorded two cases, one of degeneration of the corpus striatum, another of echinococcus—cysts in it which caused an elevation of temperature. These cases are very clear, and undoubtedly seem to show the corpus striatum to be a thermic centre.

Thalamus Opticus.—The cases bearing on this point are not very definite.

Subnormal Temperatures.—There has been recorded, one by Dr. Farnham of Cambridge, a case of typhoid whose preceding high temperature was succeeded by a fall to 95.4° F. In my experiments upon the heat centres it was found that the elevated temperature was succeeded by a fall below normal, and heat production also corresponded to it. Dr. Williams also found the temperature in phthisis characterized by an afternoon pyrexia and a subnormal temperature (94° F.) in the morning.

In the convulsive affections I have shown that tetanus mainly causes heat through the stimulation of thermic centres, and not by muscular movement. In chorea Prof. Osler, from an analysis of 410 cases, arrived at the conclusion that fever takes place when rheumatism sets in, when endocarditis becomes severe, and in some cases of chorea insaniens. There may be the most intense and violent movements without any rise of temperature, and in a great majority of cases remain afebrile throughout.

In epilepsy, according to Lemoine, in nearly two hundred observations of isolated attacks, the average rise was 1.2° F.

Tumors and lesions of the spinal cord, oblong medulla, and pons varolii are usually followed by a high temperature due to a removal of the influence of the thermotaxic centre, permitting the spinal thermogenesis to become exaggerated.

Low temperatures are due to the excitation of the thermotaxic centres, or their fibres, or to affection of the thermolytic apparatus.

High temperatures have been reported ranging as high as 125° F. to 128° F. These cases, if true, and I am inclined to believe them possible, are paralelled by Allenberg and Landois' experiments upon the Rolandic centres in dogs, where they found a difference of 23° F. between the extremities. The rapid and high elevation of temperature often seen in malarial paroxysms indicates, to my mind, an action on the thalmic heat-centre. This is the only centre whose rise and fall of temperature corresponds in time and degree to that seen in these conditions.

Cases of temperature of 110° in children have been reported which lasted for about an hour and then fell to normal, the patients recovering. In the several hundred of temperature experiments upon animals, in only one case have I seen the preceeding cases paralelled. Thus, after a grain and a half of atropin per jugular,the temperature sank to 97° F., but on applying sciatic irritation the temperature rose rapidly to 102° F., a gain of five degrees in sixteen minutes. It is well known that a dose of atropin of this size would prevent the sensory irritation from having much effect upon the vaso-motor apparatus in producing contraction of the vessels. Atropin in large doses paralyzes the main vaso-motor centre.

Diagnostic Value.—If the symptoms point to a lesion of the pons certainly high temperature will be an important aid. If the symptoms indicate a lesion about the corpus striatum then high temperature with general muscular wasting will be strong corroboration of the diagnosis.

These points will be either confirmed or refuted by lesions in man, and I hope this paper will stimulate neurologists to make more frequent observations upon temperature.

DISCUSSION ON DR. OTT'S PAPER.

Dr. DERCUM introduced Dr. Hare, who referred to the experiments of Dr. Ott. The fact that destruction of a centre caused elevation of temperature could not be said to prove that the centre in question was a heat centre, but rather that it was an inhibitory heat centre; its function was to inhibit rather than to stimulate heat production. There was an apparent fallacy, too, in the statement in regard to atropin. Atropin does not paralyze the vaso-motor centres. The chief action of atropine is upon the motor rather than upon the sensory system.

Dr. GRAY referred to a case of leptomeningitis above the fissure of Sylvius, in which the temperature was at no time above the normal.

Dr. LLOYD referred to a case of subnormal temperature after injury of the cortical motor region.

Dr. OTT explained that he had stated simply that destructive excision of the centres caused a rise of temperature; he had not stated whether those centres were inhibitory or not. In regard to atropin, he had stated that atropin inhibited the vaso-motor reflexes. The subnormal temperature in Dr. Lloyd's case would be explained by irritation of that region of the cortex, by the removal of which temperature was increased. It was not usual to observe the temperature of patients suffering with nervous disease, and variations might be overlooked. The fact that lesions about the fissure of Rolando were not always followed by an elevation of temperature proved nothing one way or the other. Paralysis also did not always result.

Dr. E. D. FISHER, of New York, then read a paper entitled:

CLINICAL REPORT OF CASES OF EPILEPSY FOLLOWING CEREBRAL HEMIPLEGIA.

Of late much has been written in regard to cerebral hemiplegia in children, or cerebrale kinderlähmung, both in this country, in England, and on the continent. In recording the following cases I would, however, lay particular stress on the epileptic seizures associated with the disease. The picture of these cases has been so often drawn that it has become a familiar one. The hemiplegia unilateral and bilateral, as it is sometimes termed, coming on at birth, or in the first few years of life, is similar in most respects to that of adult hemiplegia, with the addi-